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Case Report

First case of human spondylodiscitis due to *Shewanella algae*

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SUMMARY

We present the first case of human spondylodiscitis due to *Shewanella algae*. Our patient did not have any predisposing factors. The portal of entry was probably a cutaneous lesion on the leg, exposed to seawater. Bacteria were isolated in pure culture from a needle biopsy specimen of the vertebral disk. Automated identification systems identified the organism as *Shewanella putrefaciens*. However, molecular biology identified it as *S. algae*. Treatment with ceftriaxone and amikacin, then ciprofloxacin successfully addressed the infection. We also review four published cases of human osteoarticular infections caused by *Shewanella spp*: two cases of arthritis and two cases of osteomyelitis. Two patients had predisposing factors, and contact with water was found in two cases. The clinical, radiological and biological characteristics of *S. algae* spondylodiscitis are indistinguishable from those of spondylodiscitis of other causes. A cutaneous lesion with exposure to water is a potential portal of entry. Molecular typing is necessary to obtain a precise bacteriological identification.

1. Introduction

The bacterium *Shewanella spp*, first isolated in 1931 from putrefied butter, was originally classified as *Achromobacter putrefaciens*.[1] In 1941 it was reclassified under the name of *Pseudomonas putrefaciens* on the basis of morphology.[2] The first human isolate was described in 1964.[3] In 1972 it was reclassified as *Alteromonas putrefaciens* on the basis of its G+C content.[4] In 1985 it was yet again reclassified in a new genus *Shewanella* on the basis of comparative 5S rRNA sequences, the type species being *Shewanella putrefaciens*.[5] *Shewanella alga* was recognized as a new species in 1992[6] and renamed *Shewanella algae* in 1997.[7] More than 30 species of *Shewanella* have been described but the only *Shewanella spp* that have been recovered from human infections are *S. putrefaciens* and *S. algae*. *Shewanella sp* is a saprophyte widely distributed in nature worldwide. It is mainly found in marine environments, but it can also be isolated from all kinds of water reservoirs (lakes, rivers, open wells, sewage), soil, oil emulsions, fish, beef, poultry, and dairy products.

We report here the first case of spondylodiscitis caused by *S. algae* in the human. Our patient did not have any predisposing factors. The portal of entry was probably a cutaneous lesion on the leg, exposed to seawater. The bacterium was isolated in pure culture from a needle biopsy specimen of the vertebral disk. Automated identification systems misidentified the pathogen as *S. putrefaciens*. It was identified as *S. algae* by 16S rRNA sequence analysis.

We performed a PubMed search for reports on *Shewanella spp* osteoarticular infections and we summarize the four cases reported so far.

2. Case report

A 58-year-old man was admitted to the University Affiliated Hospital of Amiens, France, in August 2007 with severe back pain after moving his furniture two days earlier. His medical history was characterized by obesity, hypertension, and gout. On admission he had a body temperature of 38.2 °C. Medical examination revealed spinal stiffness and a 4 × 2 cm cutaneous–subcutaneous lesion on the left leg with exudative discharge and cellulitis. Three weeks earlier, the patient had injured his leg on a metal bar. He then went fishing (water level above the knee) in the Channel at Equien, France. As his cutaneous lesion was not healing he was treated for six days with oral pristinamycin (1 g twice daily) by his general practitioner. Further examinations, including a neurological examination, showed no other abnormalities. There was no peripheral adenopathy. Biological investigations showed a serum level of C-reactive protein (CRP) of 205 mg/l (normal <5 mg/l) and an erythrocyte sedimentation rate (ESR) of 67 mm/h (normal <10 mm/h). A blood leukocyte count and blood chemistry were normal. A lumbar computed tomography (CT) scan showed only degenerative disk disease. Echocardiography was normal. Three sets of blood cultures (BacT/Alert, BioMérieux, Marcy l'Etoile, France) were collected and remained sterile after five days.

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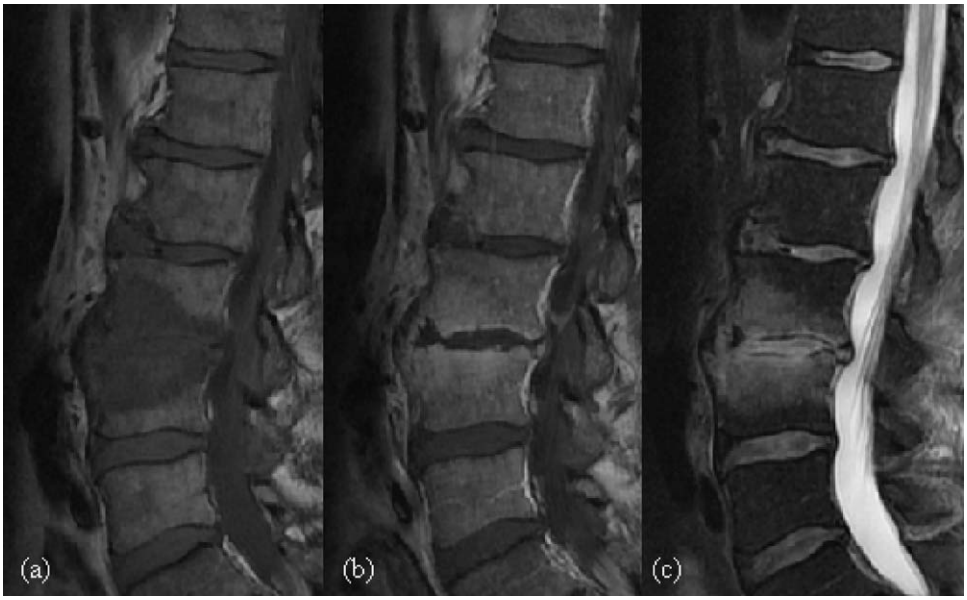

Figure 1. Sagittal magnetic resonance images of the lumbar spine. (a) T1-weighted image demonstrating low signal in the L3–L4 disk and adjacent vertebral bodies with destruction of the superior endplate of L3 and inferior endplate of L4. (b) T1-weighted post-gadolinium image showing enhancement of the L3 and L4 vertebral bodies and the anterior paraspinal soft tissue. (c) T2-weighted image demonstrating increased signal intensity in the L3–L4 disk and the L3 and L4 vertebral bodies.

Unfortunately, the lesion exudate was not cultured. The patient's pain decreased after rest and with the administration of analgesics. The cellulitis improved with prolonged prescription of pristinamycin for a total duration of two weeks. Laboratory tests demonstrated a decrease in inflammatory reactants (CRP of 27 mg/l, ESR of 42 mm/h). The patient was discharged to local care two weeks after his admission.

Three days later, he was readmitted for progressive lower back pain. Magnetic resonance imaging (MRI) of the dorsolumbar rachis showed typical septic spondylodiscitis at L3–L4 (Figure 1). A needle biopsy of the vertebral disk was performed under sterile conditions using CT guidance. The aspirated material was sent for microbiological analysis. Microscopic examination of direct Gram-stained smears revealed polynuclear cells and a few Gram-negative rods. Samples of the aspirated material were inoculated onto a chocolate agar plate (BioMérieux) incubated at 37 °C in a 5% CO_2 atmosphere and onto 5% sheep blood agar plates (BioMérieux) incubated at 37 °C under aerobic and anaerobic conditions. A brain–heart infusion broth (Oxoid, Basingstoke, Hampshire, UK) was inoculated and incubated at 37 °C. Fungal and mycobacterial cultures were performed. After 48 h, pure culture of a few large mucoid colonies with a salmon-pink color was observed on the chocolate agar plate incubated under CO_2 atmosphere and on the sheep blood agar plate incubated under aerobic conditions. Bacteria were isolated from the brain–heart infusion broth. Colonies exhibited β-hemolysis on sheep blood agar. No growth was observed on the sheep blood agar plate incubated in an anaerobic atmosphere. Oxidase and catalase tests were positive. Bacteria were motile and Gram-negative.

Biochemical identification systems ID32E, ID32GN, and API20E (BioMérieux) identified the organism as *S. putrefaciens* with 99% certainty. However, the β-hemolysis on sheep blood agar, the growth at 41 °C and not at 4 °C, the growth on nutrient agar containing 6% NaCl and on Salmonella–Shigella agar, and the inability to oxidize carbohydrates, suggested that the bacteria were *S. algae*.[8] The antibiotic susceptibility pattern was determined by a disk diffusion method (BioRad, Marne la Coquette, France). The organism was susceptible to amoxicillin, ticarcillin, piperacillin, aztreonam, cefotaxime, ceftazidime, cefepime, imipenem, gentamicin, tobramycin, amikacin, colistin, trimethoprim–sulfamethoxazole, and ciprofloxacin and resistant to cephalothin and fosfomycin according to the criteria of the Comité de l'Antibiogramme de la Société Française de Microbiologie for *Pseudomonas aeruginosa*.[9] Fungal and mycobacterial cultures were negative. For formal identification of the bacterial species, the bacterium was sent to the Institut Pasteur (Paris) for molecular identification by 16S rRNA gene sequencing, where it was definitively identified as *S. algae*.

The patient was first treated intravenously with ceftriaxone (3 g/day) and amikacin (1.5 g/day) for two weeks, then orally with ciprofloxacin (1.5 g/day) for a total duration of 12 weeks. He wore a custom-made, thermoformed, resin brace for 12 weeks to immobilize his lumbar region. His pain abated and laboratory tests showed no inflammation. Three months after completion of the treatment the patient was well.

3. Discussion

To our knowledge, this is the first reported case of human spondylodiscitis caused by *S. algae*. It was isolated from a normally sterile site in pure culture. The skin defect of the leg exposed to seawater may have been the portal of entry and the vertebral disk was probably infected after an asymptomatic bacteremia.

S. algae and *S. putrefaciens* are rarely recovered from human specimens. They are usually linked to contact with seawater in countries with a warm climate or during the summer in temperate countries. Their isolation is usually indicative of colonization. They are often isolated as part of a mixed bacterial flora.[10–12] Their pathogenic role has been established in only a limited number of cases.

Commercial automated identification systems could not distinguish *S. algae* from *S. putrefaciens* because the chemical reactions used thus far fail to discriminate between these two species. Consequently, most isolates reported as *S. putrefaciens* might in fact belong to the species *S. algae*.[6,8,11,13] *S. algae* is the predominant human pathogen and an experimental study on mice has shown that it is more virulent than *S. putrefaciens*.[14]

Shewanella spp are associated with a broad range of infections in both patients with underlying diseases and in healthy patients. Fulminating disease is described in patients with severe underly-

Table 1
Published cases of *Shewanella spp* osteoarticular infections in humans

Case No. (Ref.)	Affected site (infection)	Sex (age, years)	Predisposing factors	Portal of entry	Sample type	Identification	Outcome
Our case	Lumbar rachis	M (58)	No	Cutaneous lesion of left leg in a marine environment	L_3–L_4 disk	*S. algae* (molecular identification)	Favorable
Case 1 (Pope et al.[31])	Sacroiliac joint (osteomyelitis)	F (17)	Thalassemia, splenectomy	Subcutaneous infusion	Blood	*Pseudomonas putrefaciens* (method not specified)	Favorable
Case 2 (Roger et al.[29])	Left wrist and right ankle (arthritis)	M (62)	End stage renal failure	Peritoneal dialysis	Blood	*Pseudomonas putrefaciens* (method not specified)	Favorable
Case 3 (Levy and Tessier[30])	Second proximal interphalangeal articulation of the foot (arthritis)	M (48)	No	Cellulitis of the foot after puncture by a sea urchin	Articular fluid and periarticular tissue	*Shewanella putrefaciens* (biochemical identification by API20NE)	Favorable
Case 4 (Botelho-Nevers et al.[32])	Tibia (osteomyelitis)	F (45)	No	Open fracture with contact with stagnant water	Bone and intramedullary tissue	*S. algae* (molecular identification)	Favorable

M, male; F, female.

ing debilities. The portal of entry is frequently injured skin.[15,16] The most common clinical syndrome reported in the literature is infection of the skin and the soft tissues.[10,17] It often happens in elderly patients with chronic ulcers of the lower extremities, but soft tissue infections in healthy subjects have also been described.[18,19] Bacteremia is often present, but its evolution is usually benign. Only a few patients will develop necrotizing fasciitis.[20–22] In Denmark the most common clinical syndrome is acute otitis media, which is mostly found in children after contact with seawater during particularly hot summers.[8] A cerebellar abscess following otitis media has been reported.[23] In a few patients *Shewanella sp* has been documented as a pathogen causing intra-abdominal infections,[24] lower respiratory tract infections,[12] meningitis,[25] and abscesses.[26] Severe disease with bacteremia has been described in patients with predisposing factors such as immunodeficiency,[27] malignancy,[10,20] hepato-biliary disease,[10,22] and renal failure.[11,21,28,29]

Only two cases of arthritis[29,30] and two cases of osteomyelitis[31,32] have been reported (Table 1).

Our case was a spondylodiscitis in a patient without any predisposing underlying disease. The source of infection was likely a cutaneous leg lesion, following trauma in a marine environment. The bacterium was isolated in pure culture, and automated identification systems identified it as *S. putrefaciens*, but a few phenotypic criteria were in favor of *S. algae*. This presumptive identification was confirmed by molecular biology. The outcome under prolonged treatment was favorable.

In summary, the clinical, radiological, and biological characteristics of *S. algae* spondylodiscitis were indistinguishable from those of spondylodiscitis caused by the usual organisms. We report here the first case of spondylodiscitis due to *S. algae* and this case is significant in two aspects. Firstly, it highlights the need for molecular typing of *Shewanella spp* to obtain a precise identification of the bacterium. Secondly, it raises the possibility that *S. algae* is a causative pathogen of spondylodiscitis; this is especially true in patients with cutaneous lesions and water exposure, because it is a potential portal of entry for the pathogen.

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